MASSACHUSETTS INSTITUTE OF TECHNOLOGY
DEPARTMENT OF BIOLOGY
CAMBRIDGE 39, MASSACHUSETTS

13 January 1959

Jan. 15

Packing now!

Dear Josh and Es:

Best wishes for the move!

I enclose a summary of our transduction experiments until December 15. Since then, using your strain W4032, the picture has been fully confirmed and clarified:

W4032, which according to you has a deletion involving both ß-gal and permease, behaves like Shigella: it gives heterogenotes, which give HFT lysates when superinfected. In addition, the various "P1-Lac$^+$" phages derived independently have specific transduction ratios on various recipients.

It becomes essential to define exactly the nature of the W4032 deletion; if you know more about it, let me know.

W4047 works well as recipient but reverts at a rate of about 10^{-8}-10^{-9}. If you have several stable Lac$^-$ (not permease -, see below) I'll ask for them when convenient.

W4056 is nontransducible. I suspect it may have a suppressor.

I look forward to seeing you in Berkeley.

The same!

Best regards,

S.E. Luria/ GR.

S. E. Luria

SEL:gr

working hard on it! Then presumably it is the interference with crossing over to yield lac$^+$ which either induces heterogenosis, or heavenly rare heterogenotes as visible Lac$^+$. Which is it ?

Joshua.

1. Transduction by phage P1 among strains of E. coli K12 or B, for all markers tested, gives recipient cells which, after selection, are all phage sensitive and stable for the transduced characters (provided multiple infection or superinfection is avoided). This includes transduction of Lac^+ into various Lac^- coli strains.

2. The same is true for transduction from Shigella into coli, including transduction of Lac^+ from Shigella donors that are stable Lac^+ as a result of mating with E. coli.

3. Transduction from E. coli into Shigella gives results varying with the marker. Some markers behave as in item 1 above (recipients all P1 sensitive); others give mostly lysogenic recipients. The critical result is obtained with Lac^+ (irrespective of donor, either coli or Shigella-coli hybrid): All the Shigella $^{ABS}_{\lambda}$ Lac^+ unstable heterogenotes. These fall into various categories with respect to P1: the majority (types 1 and 4) are defective lysogenic, segregating P1-sensitive Lac^-; others (type 7) are lysogenic, unstable, segregating P1 lysogenic Lac^- and P1 defective Lac^+ (type 7 is presumably a double carrier, for P1 and P1 defective).

4. Type 4 is semistable (frequency of Lac^- segregation about 10^{-3} per generation). When superinfected with P1, it segregates P1 lysogenic Lac^-, P1 defective Lac^+, and rare P1 sensitive Lac^-.

5. When type 4 is irradiated (maximum induction 5%) and superinfected with P1 grown on Lac^-, the lysate is HFT for Lac^+ (ratios "Lac^+ transduction/active P1" up to 5% instead of 10^{-7} for LFT). Note: these lysates are HFT both for E. coli and Shigella at equal frequency (see item 6, however). The coli recipients are Lac^+ stable P1 sensitive; the Shigella recipients are again heterogenotes, mainly type 4.

6. HFT transduction into Shigella requires help from active phage (1 active P1 per cell). HFT transduction into coli does not require help.

7. We conclude at this point that the heterogenotes carry a "P1 def-Lac^+" (P1-Lac) element that can reproduce as such in Shigella. There is evidence for a variety of P1-Lac elements from two sources:

a) Different rare heterogenotes types (types 5 and 7) give rise to lysates that transduce at medium-low frequency (MFT) and give rise to heterogenotes with different degrees of immunity to P1.

b) Some HFT lysates do not transduce into certain E. coli Lac^- mutants, while others do (ratios over 10^{-4}). They all transduce into Shigella.

8. We now postulate that a P1-Lac element, when entered into Sh, cannot "donate" the Lac^+ character and must reproduce as such. When a P1-Lac enters a coli cell, it can donate Lac^+, whether it multiplies or not (hence, the lack of help requirement). Evidence for this is obtained from UV experiments (item 9).

9. When an HFT lysate is irradiated, the transducing ability for E. coli decays very slowly; the transducing ability for Shigella decays fast (slopes for P1 activity, Shigella transduction, coli transduction approximately 100: 30: 1). We interpret this as expressing the requirement for a hit within a restricted recombining region for transduction into coli versus the requirement for a hit overpassing multiplication of the exogenote for transduction into Shigella. (Reactivation, the role of the helping phage, and of the host cells are being studied.)

10. Generalizing:

a) We propose that all transduction is mediated by genetic elements arising by "recombination" between phage and cell chromosome. If these elements are defective and multiply poorly, but can donate their host character, selection for transduced cells eliminates the heterogenotes and yields only stable P1 sensitive transductees. Only when the host character cannot be donated will selection yield heterogenotes.

b) Exogenotes that can neither multiply nor donate will give abortive transduction. The carrier cells may be immune or not depending on whether the exogenote contains an effective immunity gene. A whole series of elements may exist intermediate between this case and that of P1-Lac.

c) λ-Gal appears to be an exogenote that can donate (rarely) and multiplies well enough to be found after selection.

d) A combined element may be formed without losing its "phage-genes." This may give all lysogenic transductions (barring prophage or preprophage segregation after donation of the host gene). In a recipient that cannot accept the phage-carried host genes, these elements will behave as "converting phages." (Note that phage ε^{15} of Salmonella converts, reversibly, antigen 10 to antigen 15, but strains are found with antigen 15, without phage ε^{15} and resistant to it).